Vaccination against smallpox

SIR,—On 26 August the Chief Medical Officer and Chief Nursing Officer at the Department of Health and Social Security sent a letter to all doctors telling them that, following the declaration by the World Health Assembly on 8 May 1980 that the world is now free of smallpox, the Joint Committee on Vaccination and Immunisation had recommended that routine vaccination against smallpox should be provided only for people working with smallpox virus and staff who have agreed to work with patients strongly suspected of having smallpox together with, conditionally, their families and those who are engaged in the manufacture of the vaccine or who perform vaccination.

The letter from the CMO and the CNO goes on to remind doctors that there is no medical reason for vaccination and that the procedure carries a small but recognised hazard. It recognises that some countries still officially require valid international certificates against smallpox and that some embassies will not issue visas until a valid international certificate has been produced. It finishes by saying that doctors "will therefore wish to satisfy themselves that the traveller requesting vaccination fully understands the position."

The doctors practising in my district probably are required to immunise more travellers than doctors in any other comparable area in this country. There has therefore been great interest in the contents of the CMO's letter. One of the practitioners in the City wrote to both the medical defence societies and received replies which I consider will be of the greatest interest to doctors throughout the country. In short, the defence societies now take the view that, if a doctor carries out a vaccination against smallpox and complications occur, the patient could allege that the doctor had carried out a procedure which was medically contraindicated, and further that it would be difficult to formulate a defence against such an allegation. The societies go on to say that it is their preference that doctors should refuse to vaccinate except in those cases set out in the letter from the CMO, but should as an alternative provide a certificate to say that vaccination is contraindicated.

It is important that doctors should realise that there are two different recommendations being made here, in that the CMO appears to consider that vaccination against smallpox is a matter for the personal opinion of the doctor, provided that he ensures that the traveller fully understands the position. The medical defence societies, on the other hand, say that doctors should refuse to vaccinate anyone. They do not specifically say that they will not cover doctors who insist on vaccinating but it appears to me, as to other doctors with whom I have discussed the matter, that the possibility cannot be ignored. The result has been a unanimous decision that it would be foolhardy to continue to vaccinate, whatever the clinical pros and cons of the matter.

Two questions emerge from all this: (1) Is it the proper function of the medical defence societies so to influence individual doctors in their clinical judgment? (2) If it is proper, should the defence societies not circulate all doctors who subscribe to them, setting out their decisions and the reasons for them?

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A better system for polio vaccination in developing countries?

SIR,—Dr Dion Bell in his comment on the eventual reasons for a low potency of polio vaccine stated (20 September, p 810), "It is well known that freezing polio vaccine largely inactivates it." To support his opinion, he refers to a statement by Wellcome that "freezing is harmful to the vaccine." It has to be stressed that in a recent investigation concerning this aspect of adverse storage on polio and other live vaccines Wellcome came to the conclusion that the consequences of such a storage "are not likely to be serious."[1] Lundbeck in a study of the same problem stated, "Freeze-dried and liquid living virus vaccines are not significantly reduced in immunising potency by freezing and thawing as many as five to ten times."[2]

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[1] Finter NB, Ferris R, Kelly A, *et al. Develop Biol Standard* 1978;**41**:271-6.
[2] Lundbeck H. *Weekly Epidemiological Record* 1977; **52**:119.

The coronary care controversy

SIR,—I sincerely hope the excellent review of coronary care by Drs J M Rawles and A C F Kenmure (20 September, p 783) will jolt the profession and other relevant parties out of the unnatural vacuum of inactivity of recent years.

There has been an illogical and emotional overshoot to the Nottingham and Teeside survey results, with sweeping generalisations and condemnations of mobile coronary care and even the coronary care unit. Under critical analysis these surveys have never really tolled the death bell of coronary care but have simply served to demonstrate two basic truths. Firstly, there are great difficulties in studying the acute coronary attack and obtaining a suitable model for study with meaningful randomisations. Secondly, they confirm that coronary care is firmly a community problem and an early problem. From the latter view point as a family doctor interested in coronary care, I am painfully aware of the consequences of leaving the early coronary care patient at home, as is suggested or implied by the results of these surveys. The practical difficulties of diagnosis, adequate analgesia, and management of complications cannot be underestimated.

The treatment of premature electrical death in the early hours after infarction is what coronary care is all about and has proved to be a reasonable and effective exercise associated with good long-term survival. It is impractical for the majority of general practitioners to offer their patients defibrillation, be consistently available for the problem patient requiring repeated analgesia, and attend within minutes of an acute deterioration. Furthermore, the individual GP's experience of the early attack is relatively infrequent and inconsistent with the aquisition of expertise in treatment and emergency resuscitation, and is inappropriate in the home anyway.

In the final analysis, as doctors where would we want to be for our own coronary attack? On the assumption that we would take action fairly early, how many of us would retire to bed after analgesia by his family doctor, who promises to revisit some time later? The answer is very few, I think—we would all be tucked up in our local coronary care unit just in case, as our patients should be. The early coronary patients should be stabilised by a mobile coronary team and transferred to a unit where expertise is concentrated.

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Atypical angina

SIR,—The reference of anginal pain exclusively to the left thenar eminence by Dr A Lahiri and his colleagues (20 September, p 782) is interesting in that there was no pain in the thumb. It is appreciated that the radial side of the hand is supplied by the C6 dermatome but never, so far as I know, has anginal pain been experienced in the thumb.[1 2] The papers by Sampson and Cheitlin[3] and McKenzie[4] do not differentiate between the thumb and the thenar eminence. Indeed, palor, sweating, and tingling in the area to which pain is referred invariably spare the thumb. The autonomic manifestations of "cardiac" pain are protean and hence it is quite useful in diagnosis to be aware of an area where none of these is experienced.

Bilateral finger, thenar eminence, forearm, and arm pain, especially if aggravated by exercise or anxiety, are almost exclusive to causes originating in the heart—the exception being disease, whatever the cause, in the mediastinum. Unilateral hand and arm pain may, of course, arise from cervical spondylitis and disorders of the shoulder or brachial plexus and the carpal tunnel and are not related to exertion. Thrombotic or embolic occlusion of the brachial artery will cause arm claudication and the pain will involve the forearm and the whole hand. Localised skeletal or musculofascial disorders of the hand should not enter into the differential diagnosis of angina.

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[1] Daley R. *Br Med J* 1957;ii:173-9.
[2] Daley R. *Proc R Soc Med* 1960;**53**:26-8.
[3] Sampson JJ, Cheitlin MD. *Prog Cardiovasc Dis* 1971;**13**:507-31.
[4] McKenzie J. *Angina pectoris*. London: H Frowd and Hodder and Stoughton, 1923-31. (Oxford Medical Publications.)

Vomiting as a diagnostic aid in acute ischaemic cardiac pain

SIR,—In their recent interesting article (6 September, p 636), Dr D A Ingram and others stated in the final paragraph, "Although our findings fail to explain the cause of vomiting after myocardial infarction, the clear-cut relation with transmural necrosis suggests that the vomiting reflex, if such exists, might arise as a consequence of damage to the subpericardial tissue." The work of Abrahamsson and Thorén and others may help to elucidate this reflex.

Ventricular receptors with non-myelinated C fibre vagal afferents are found throughout the wall of the left ventricle[1] and are stimulated by mechanical and irritant events,[2] particularly distension and raised left ventricular end diastolic pressure.[3] Their reflex effects include gastric dilatation and vomiting,[4] cardiac slowing, hypotension, and a tonic effect on renin release.[5] These ventricular C fibre vagal afferents are probably also responsible for the